

Atrial Flutter Associated With Acute Myocardial Infarction

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Between 1966 and 1968, 35 of 340 patients (10.3 percent) admitted to the Los Angeles County-University of Southern California Medical Center with documented myocardial infarctions were noted to have atrial flutter while being monitored in the coronary care unit. This incidence is higher than that found in most previous studies.

Patients with atrial flutter had significantly higher mortality than infarction patients without flutter ($P < 0.05$). The increased mortality for those with flutter was apparently the result of concurrent complications and probably not because of the arrhythmia alone.

Atrial flutter recurring for more than one day was associated with further increase in mortality ($P < 0.02$). Also, there was a higher death rate among patients with flutter who had anterior infarctions than among those with inferior infarctions, but the difference was not statistically significant.

Most patients responded to treatment with antiarrhythmic drugs, but prior digitalis therapy apparently did not prevent the onset of atrial flutter.

THE USE OF continuous electrocardiographic (ECG) monitoring systems in coronary care units has made it possible to observe and evaluate the rhythm changes associated with acute myocardial infarction (MI). Because of the acute prognostic significance of ventricular arrhythmias, attention is usually centered on this arrhythmia. Several studies have shown that supraventricular arrhythmias are a common occurrence in the setting of acute myocardial infarction and may have clinical importance.¹⁻⁴

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The genesis and prognostic significance of supraventricular arrhythmias complicating myocardial infarctions warrants further study. Suggested causes of atrial tachycardias include congestive heart failure and cardiogenic shock, sinoatrial node ischemia and infarction, pericarditis, atrial infarction or ischemia, and vagal reflexes or increased sympathoadrenal activity.^{2,5-11} Most authors feel that the supraventricular arrhythmias alone do not increase mortality but that the underlying process causing the arrhythmias may be associated with decreased survival.^{1,2,4}

Few studies have been concerned specifically with the occurrence of atrial flutter in the setting of acute myocardial infarction. We have, there-

ABBREVIATIONS USED IN TEXT

AV=atrioventricular
CPK=creatine phosphokinase
ECG=electrocardiogram
MI=myocardial infarction

fore, evaluated this arrhythmia in the coronary care unit with regard to incidence, mortality, location of infarction, time of onset and recurrence of the arrhythmia, treatment and associated complications in patients with a diagnosis of definite or probable infarction.

Patients and Materials

There were 35 patients with atrial flutter among the 340 patients with infarction admitted to the Los Angeles County-University of Southern California Medical Center, Coronary Care Unit in two years. Patients were predominantly from the lower socioeconomic population in the Los Angeles area and their admission to the unit was on a bed-available basis. The study involved 22 men and 13 women, ranging in age from 43 to 77 years and 55 to 80 years, respectively.

The diagnosis of myocardial infarction was confirmed on the basis of a characteristic history, typical ECG changes and elevated levels of serum aspartate aminotransferase and creatine phosphokinase (CPK).¹² CPK fractionation was not available during the two-year study. Congestive heart failure was diagnosed on the basis of x-ray findings (cardiomegaly, differential blood flow to the upper lobes, vascular congestion and Kerley's lines), distended neck veins, hepatomegaly, persistent rales, gallop rhythm or increased central venous pressure. Shock was evident when there was a systolic blood pressure less than 80 mm of mercury, decreased or absent peripheral pulses, cold and clammy extremities, cyanosis and oliguria.

On admission a 12-lead ECG was taken and patients were monitored by cardiac nurses using oscilloscopes at bedside and central stations with a five-lead attachment, rate alarms and tape delay loops.¹³ The patients were kept at bedrest, with a low-salt diet, oxygen, sedation and diuretics administered as required. The patients were observed for a minimum of five days after admission, and longer in those cases in which the patient's condition was unstable, followed by progressive ambulation per protocol.¹⁴

TABLE 1.—Time of Onset of Atrial Flutter Following Myocardial Infarction in 35 Patients

Time of Onset (days)	Number of Patients		
	Those Who Lived	Those Who Died	Total
0	2	0	2
1	6	1	7
2	0	2	2
3	6	3	9
4	5	1	6
5-14	6	3	9
TOTAL ...	25	10	35

TABLE 2.—Persistence of Episodes of Atrial Flutter Following Myocardial Infarction in 35 Patients

Length of Flutter Episode	Number of Patients With Episodes		
	Who Lived	Who Died	Total
Minutes	3	0	3
Less than a day	7	2	9
A day	9	1	10
	19*	3*	22
One to two days	6	2	8
More than two days	0	5	5
	6*	7*	13
TOTAL	25	10	35

* $\chi^2 = 4.65$; $P < 0.02$.

Results

Incidence and Mortality

Of the 340 patients admitted to the coronary care unit from 1966 to 1968, 35 (10.3 percent) were noted to have episodes of atrial flutter. Ten of the 35 patients with atrial flutter died (29 percent). This is a significantly higher death rate than that for all myocardial infarction patients during the same period (15.5 percent, $P < 0.05$).

Location of Infarction

In the 35 patients with atrial flutter, 16 had anterior MI's, nine had inferior MI's and one had a posterior MI. In the remaining patients, the locations were combined or undetermined.

Of the 16 patients with anterior infarctions, 7 died (44 percent), whereas only 1 of 9 patients with inferior infarctions died (11 percent). Although this difference is not statistically significant, the tendency toward increased mortality in patients with anterior infarctions is evident.

Time of Onset

In seven patients atrial flutter developed the day after infarction, in two it occurred two days

after infarction, in nine it developed on the third day and in six it occurred on the fourth day. In two patients with no previous history of arrhythmias, the flutter was noted on admittance to hospital. In the other patients it developed between the 5th and 14th day. Table 1 shows the time of onset of flutter for the patients who lived and those who died. There was no significant difference in onset of flutter between these populations.

Persistence and Recurrence

In our series, three patients had episodes of flutter lasting only minutes. In nine they lasted for hours (but less than a day) and in ten the episodes occurred intermittently for 24 hours (see Table 2). Monitoring also showed that in eight patients the atrial flutter lasted for one to two days, in five it lasted for more than two days and in two the episodes lasted for up to 10 days. Substantially more patients in whom the flutter lasted for more than a day died than those with shorter episodes. Although patients with high CPK values tended to have a poorer prognosis, there was no significant association between high CPK values and occurrence or persistence of atrial flutter. An example of a short episode of atrial flutter is shown in Figure 1. Onset is with a premature beat, a high degree of atrioventricular (AV) block is present, and the sinus node recovery time exceeds the normal R-R interval.

Treatment

Of the 35 patients with atrial flutter, 22 were already receiving digitalis before the onset of flutter. In almost every case prior digitalis therapy had been instituted because of symptomatic congestive heart failure. When compared with the 13 patients not receiving digitalis, we found that the

patients treated with this drug tended to have higher degrees of atrioventricular (AV) block and slower ventricular responses; however, these differences were not statistically significant. Seven of the ten patients who subsequently died were receiving digitalis at the time of the onset of atrial flutter. Thus, previous administration of digitalis did not significantly affect mortality.

Eight patients who were not previously treated with digitalis were given this medication at the onset of atrial flutter. Nineteen patients who were not receiving quinidine before the onset of flutter were given this medication. One patient was cardioverted and seven were treated supportively. Twelve patients had been taking either procainamide or lidocaine before the onset of atrial flutter. No adverse effects of antiarrhythmic therapy were noted in the patients with atrial flutter.

Only three of the 35 patients with atrial flutter were receiving anticoagulant drugs. One of these three subsequently died of myocardial failure. No relationship between anticoagulation and mortality was discernible.

Previous Medical Problems and Complications

Thirteen patients had diabetes mellitus and 14 had evidence of or had a history of hypertension. Six patients (one each) had a history of bleeding peptic ulcer, gout, lues, myasthenia gravis, bronchiectasis and cirrhosis. Two had had previous cerebral vascular accidents. These problems were stable at time of onset of atrial flutter. Two patients had recovered from previous myocardial infarctions.

While in hospital for acute MI, one patient had a cerebral embolism, which occurred on the eighth day of illness while the patient was in sinus rhythm; atrial flutter, however, lasted intermit-

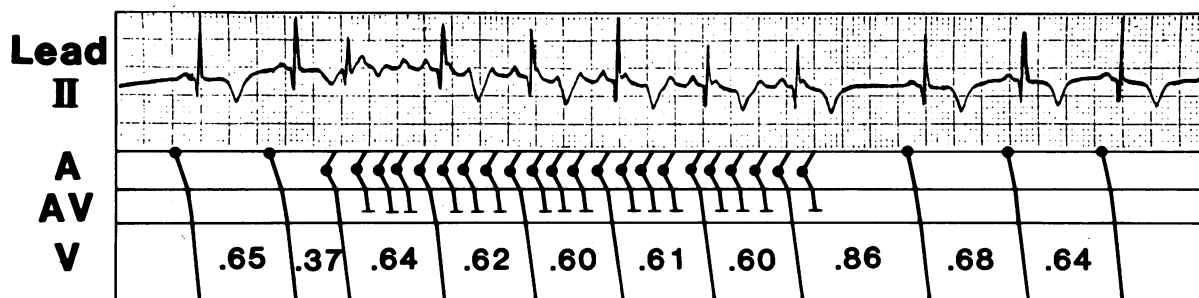


Figure 1.—Example of intermittent atrial flutter which occurred in a 55-year-old man with an inferior myocardial infarction, who had hypertension but was not receiving digitalis. The arrhythmia is initiated by a premature atrial complex, and every fourth beat is conducted to the ventricles (4-1 AV block). Spontaneous termination occurs, followed by a sinus escape interval that exceeds the normal sinus interval (860 versus 680 msec); the effective ventricular rates are similar due to the high degree of block.

tently for ten days and was treated with digitalis. Another embolic or thrombotic episode occurred on the 13th day postinfarction in this patient (see patient 5, Table 3).

Two patients had evidence of pericarditis on admission, but there were no episodes of myocardial failure or atrial infarction before the onset of atrial flutter. There was no evidence of congestive heart failure occurring secondary to the onset of atrial flutter alone, in our best judgment. Subjective shortness of breath occurred in one patient and transiently decreased blood pressure in another. There were no known recurrences after one day; if the flutter stopped for 24 hours, it did not recur.

Of the ten patients who died, autopsies were done on eight (80 percent). Recent or acute MI was found in all eight patients, with papillary muscle involvement and an intracardiac thrombus in two. Causes of death, location of infarct, and related findings are given in Table 3.

Discussion

Incidence

We confirmed 35 cases of atrial flutter associated with 340 cases of acute myocardial infarction (10.3 percent). Table 4 depicts the incidence and mortality found in other studies both before and after continuous ECG monitoring systems were developed. In the premonitoring era before 1964, the incidence of atrial flutter associated with myo-

cardial infarction ranged from 0.5 percent to 3 percent with an average incidence of 1.2 percent (46 of 3,782 cases). In the continuously monitored patients, the incidence of atrial flutter ranged from 1 percent to 6 percent, with an average incidence of 3.2 percent (59 of 1,870 cases). The incidence in our study of 10.3 percent is higher than that in most previous reports. More recent data from our coronary care unit have shown the incidence of atrial flutter in patients with infarctions to be 6 percent.¹⁵

As we expected, with the advent of continuous monitoring systems, an increased incidence of documented atrial flutter was demonstrable. Comparison of our data with Askey's study¹⁶ in the premonitored era, both of which were done at the Los Angeles County-University of Southern California Medical Center, shows almost a sevenfold increase in the incidence of documented atrial flutter. That this increase was not found with atrial fibrillation could be attributable to two main considerations.⁴ First, because of its irregular rhythm and pulse deficit, atrial fibrillation has been easily diagnosed without the aid of a rhythm strip. Second, the early onset of atrial fibrillation after myocardial infarction is more easily recognized on the admission ECG than atrial flutter, when the classic "sawtooth baseline" is lacking.

Mortality

We found that 10 of 35 patients (29 percent) with atrial flutter died, while the average mor-

TABLE 3.—*Pathological Findings at Autopsy*

Patient No.	Cause of Death	MI Location*	Coronary Artery Disease†	Associated Findings
1 ..	Perforation of left ventricle, AMI ...	Ant, Ap, S	Circ, RCA—moderate	Multiple renal cysts, PC
2 ..	Septal perforation, AMI	Inf, S	LAD—severe	Abdominal aortic aneurysm, splenic hematoma, PC
3 ..	AMI	Ant, Post, S, Pap	LM, RCA—severe	Intracardiac thrombus, pericarditis, bleeding ulcer, cecal adenoma, cerebral atrophy, PC
4 ..	AMI	Ant, Lat	LAD—severe	PC
5 ..	AMI	Ant, Ap, S	LAD, Circ, RCA—severe	Multiple cerebral infarcts, renal artery thrombus, renal papillary necrosis, PC
6 ..	AMI	Ant, Ap, S, Lat	LAD, Circ, RCA—moderate	PC, old contusion of right temporal lobe
7 ..	AMI	Post, Lat, S, Pap	LAD, Circ—severe	Intracardiac thrombus, diabetes mellitus, bladder hemorrhage
8 ..	AMI	Ant, Ap, Post, S	LAD, Circ—severe	Diabetes mellitus, PC

AMI=acute myocardial infarction; Ant=anterior; Ap=apical; S=septal; Inf=inferior; Post=posterior; Pap=papillary muscles; Circ=circumflex; RCA=right coronary artery; LAD=left anterior descending; LM=left main; MI=myocardial infarction; PC=pulmonary congestion.

*All MI's listed occurred at last hospital stay.

†Moderate=60%-80% occlusion; Severe=80%-100% occlusion.

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tality for all myocardial infarction patients during the same period was 15.5 percent. Previous studies (Table 4) have shown death rates ranging from 0 percent to 75 percent. In studies after 1964, the mortality ranged from 0 percent to 44 percent, with an average mortality in patients with atrial flutter of 36 percent. The study by Jewitt and co-workers¹⁷ shows no mortality associated with atrial flutter, although his small sample population precludes strong conclusions. Stock and colleagues¹⁸ conclude that the prognostic significance of arrhythmias in the setting of myocardial infarctions can be assessed only if one takes into account the hemodynamic status of the patient. They state that in the presence of mild infarction or cardiogenic shock, arrhythmias have little prognostic significance; however, in between these extremes arrhythmias may compromise the patient's hemodynamic state, and thus affect mortality. The study by Liberthson and associates² indicated that there was increased mortality in patients with atrial flutter compared with patients with other atrial tachyarrhythmias, but the increase was not statistically significant. Liberthson concludes that "there was no significant difference in mortality related to the presence or absence of atrial tachyarrhythmias." Our study indicated sig-

nificantly increased mortality over the general population of myocardial infarction patients ($P < 0.05$). Lown and colleagues classify all atrial tachyarrhythmias as "arrhythmias of pump failure" and they state that "patients in this group have a poor prognosis. Mortality is twice that in the rest of the population."⁶ The study by Mintz and co-workers¹⁹ in the premonitoring era also showed a statistically significant increase in the death rate associated with atrial flutter in myocardial infarction patients ($P < 0.05$). The significant increases in mortality observed in the studies by Cristal and co-workers¹ and Stock and colleagues¹⁸ involved patients with atrial flutter and other supraventricular tachyarrhythmias and so comparisons with our data are not valid.^{1,18}

Location of Infarction

Table 5 compares infarction location in the present series with that in other patients as described by other investigators. All studies show a slight predominance of anterior over inferior infarctions, but this difference was not statistically significant. Lofmark and Orinius³ stated specifically that the infarction site for those with supraventricular tachycardia was no different than for the general MI population. James⁸ has shown that

TABLE 4.—Incidence of Atrial Flutter and Increased Mortality Associated With Myocardial Infarction in Studies Before and After Development of ECG Monitoring

	Cases	Flutter	Percent	All MI Percent†	Flutter Percent‡	Increased Mortality
Studies using ECG monitoring						
Kurland ²³ (1965) . . .	16	1	6.0
Meltzer ²⁰ (1966) . . .	141	3	2.0
Jewitt ¹⁷ (1967)	222	2	1.0	24.0	0.0	No
Stock ¹⁸ (1967)	200	18*	9.0	30.0	61.0*	Yes
Lown ⁶ (1967)	300	16	5.3	17.7	43.8	Yes
Cristal ¹ (1975)	350	39†	11.0	16.0	41.0†	Yes
Liberthson ² (1976) . .	917	29	3.0	20.5	34.5	NS
Lofmark ³ (1978)	274	8	3.0
Current series	340	35	10.3	15.5	29.0	Yes ($P < 0.05$)
Studies before ECG monitoring						
Master ²⁴ (1938)	300	3	1.0	26.0	67.0	NS
Rosenbaum ²⁶ (1941) .	208	6	3.0	33.0	66.0	NS
Mintz ¹⁹ (1947)	572	4	0.7	22.0	75.0	Yes
Askey ¹⁶ (1949)	1,247	19	1.5	51.5	63.0	NS
Goldman ²⁷ (1950) . . .	127	2	1.6
Ball ²⁸ (1955)	342	7	2.0
Johnson ²⁵ (1958) . . .	187	1	0.5
Imperial ²⁹ (1960) . . .	153	1	0.7
Begg ³⁰ (1961)	46	1	2.0
Julian ³¹ (1963)	100	2	2.0
Hurwitz ³² (1964) . . .	500+	..	1.0

ECG=electrocardiographic; NS=not significant.

*Includes all supraventricular tachycardia except atrial fibrillation.

†Includes atrial flutter and fibrillation.

‡Mortality.

atrial arrhythmias are often associated with occlusion of the artery to the sinoatrial node, with subsequent infarction with the node. Jewitt and co-workers¹⁷ point out that the sinoatrial node is supplied by the right coronary artery in 55 percent of cases and by the circumflex in 45 percent of cases. They go on to conclude that in patients with an anterior infarction, the development of an atrial arrhythmia indicates occlusion of a secondary major vessel in addition to the anterior descending branch of the left coronary artery; therefore, an atrial arrhythmia in the setting of an anterior infarction may herald an extension of the infarction.

Cristal and colleagues¹ state that the infarction site is an important determinant of the mechanism of the arrhythmia. They hypothesize that because of the coronary anatomy, the sinus and AV nodes can be impaired with relatively little myocardial damage in patients with inferior infarctions. Therefore, diverse supraventricular arrhythmias can be produced without adversely affecting the patient's prognosis. But with anterior infarctions, supraventricular arrhythmias are felt to arise because of myocardial failure or catecholamine excess, which are both related to poorer prognosis. They conclude that supraventricular arrhythmias portend a poorer prognosis in anterior but not inferior infarctions.¹ Our findings are in agreement with these conclusions—the death rate was four times greater in those patients with atrial flutter and anterior infarctions than in those with atrial flutter and inferior infarctions.

In their studies relating atrial fibrillation to myocardial infarction, neither Liem and co-workers⁹ nor McLean and colleagues¹⁰ found significant differences related to infarction site (Table 1).

Time of Onset

The figures from the present study on time from illness to onset of atrial flutter agree with those of Lofmark.³ Cristal and colleagues¹ indicate that of 39 instances of atrial flutter and fibrillation, 24 (62 percent) occurred within 24 hours of admission. However, they do not report the time of onset with respect to day the infarction occurred.

Persistence and Recurrence

In the present study, those patients with more than one day of flutter had significantly higher mortality than those with shorter episodes. There are two possible explanations for this relationship. First, persistence of atrial flutter refractory to treatment may be occurring in those with more severe heart failure and larger infarcts and, hence, increased mortality. And, second, recurrent atrial flutter may have compromised the hemodynamic status of infarcted patients and, thus, be a causative factor in their increased mortality.

In our study, the persistence of atrial flutter bore no relationship to previous digitalis therapy.

Treatment

Most of the reviewed studies favor electrical cardioversion for the treatment of atrial flutter. Jewitt and co-workers¹⁷ reported 100 percent success in converting their two patients in atrial flutter back to sinus rhythm with countershock. Meltzer and Kitchell²⁰ also stated that excellent results were obtained with countershock and that this is the method of choice for treatment of atrial flutter. Lown and associates⁵ reported that the short-acting glycoside ouabain should be used when atrial flutter is observed, but if this arrhythmia is compromising the patient's hemodynamic status, cardioversion should be done followed by

TABLE 5.—Location of Infarction in Patients With Atrial Flutter in Present Series and in Previous Studies

Study	Total No. of Cases	No. of Patients With Infarcts in These Locations				
		ANT.	INF.	POST.	Combined	Undetermined
Current series	35	16	9	1	5	4
Cristal ¹	150*	60	38	..	21	35
Kurland ²³	1	0	1	0	0	0
Jewitt ¹⁷	2	2	0	0	0	0
Lofmark ³	8	4	1
McLean ¹⁰	220†	80	70
Liem ⁹	80†	42	34	0	4	0

ANT=anterior; INF=inferior; POST=posterior.

*Patients with supraventricular arrhythmias.

†Patients with atrial fibrillation only.

digitalis and quinidine therapy to prevent recurrence. Josephson and colleagues²¹ have recommended that if these therapies fail, DC counter-shock should be used; it was successful in 95 percent of their cases. In the series of Libberthson and associates,² 20 patients with atrial flutter were treated with intravenous medications and nine were treated with DC cardioversion.

In our series, cardioversion was done in only one patient because only in this case was it felt that the persistent arrhythmia was causing hemodynamic deterioration. The other patients had either single or multiple short bursts of flutter, or intermittent flutter which did not dramatically change the hemodynamic status. Thus, pharmacologic therapy was carried out.

Haft and co-workers²² describe three cases in which atrial flutter was terminated by rapid atrial pacing and advocate this type of cardioversion. None of the previously mentioned studies have included this method, and in recent years it has had variable acceptance as the treatment of choice for atrial flutter.

Conclusions

- The incidence of documented atrial flutter after myocardial infarction was 10.3 percent in this study and has increased with the advent of continuous monitoring systems in practically all reported studies.

- There is increased mortality for infarction patients with associated atrial flutter ($P < 0.05$), but this is primarily a result of concurrent complications and probably not because of the arrhythmia alone.

- More than two thirds of the episodes of atrial flutter began three or more days after the infarction (69 percent) in our study, and similar experience has been reported elsewhere.

- When episodes of flutter persisted for more than a day, there was a significant increase in mortality ($P < 0.02$).

- Most patients responded to antiarrhythmic drugs; DC shock for cardioversion was used effectively in one patient.

- Atrial flutter in patients with anterior infarctions may indicate a worse prognosis than in those with inferior infarctions, although this difference did not reach statistical significance in our study.

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